First ihsig International symposium, March 28-29,2012 – Ghent, Belgium

The mycotoxin deoxynivalenol predisposes for the development of necrotic enteritis in broilers

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Background

Subclinical necrotic enteritis (NE) is an economically important enteric disease caused by a netB producing, Gram-positive, anaerobic bacterium, *Clostridium perfringens*. Massive intestinal proliferation of C. *perfringens* is promoted by increased availability of free amino acids. The *Fusarium* mycotoxin deoxynivalenol (DON) is a common feed contaminant with a maximum guidance level set at 5000 µg/kg feed (2006/576/EC) and may damage intestinal epithelial cells and/or their intercellular junctions, subsequently inducing protein leakage (Girish and Smith, 2008) and may thus predispose to the development of NE.

Methods

A highly reproducible *in vivo* infection model mimicking subclinical NE was used (Gholamiandehkordi et al., 2007). A total of 360 one-day-old Ross 308 broilers from a commercial hatchery were randomly divided into four groups of three replicates with 30 birds per replica. All birds were fed a starter diet during the first eight days of the experiment, subsequently a grower diet for eight days, followed by a finisher diet during the remaining days. Throughout the entire experiment, group 1 and 4 received a blank diet while group 2 and 3 received an experimentally contaminated diet with DON. All birds in group 1 and 2 were challenged orally with one ml of a culture of *C. perfringens* strain 56 containing approximately 4 x 10^8 cfu/ml for four consecutive days starting at day 17. The remaining groups received sterile medium orally. The contamination level of DON and other mycotoxins was assayed using a validated multi-mycotoxin LC-MS/MS method (Monbaliu et al., 2010).

Results

The blank feed contained DON at 75 ± 22 µg/kg (starter), 83 ± 24 µg/kg (grower) and 100 ± 29 µg/kg (finisher). The contaminated feed contained DON at 3761 ± 1100 µg/kg (starter), 4281 ± 1300 µg/kg (grower) and 4384 ± 1300 µg/kg (finisher).

At 1, 2 or 3 days after the final challenge with *C. perfringens*, chickens were euthanized and scored macroscopically for intestinal NE lesions. Chickens that received DON and *C. perfringens* had significantly (alpha=0.05, $P<0.001$) more lesions than chickens that received only *C. perfringens*, with 46.6% and 19.5% of chickens positive for NE lesions, respectively. No NE lesions were seen in the groups receiving no *C. perfringens* inoculations.

Conclusion

Feeding DON contaminated feed in concentrations lower than the maximum guidance contamination level of 5000 µg/kg to broilers is a predisposing factor for the development of NE.

References

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Acknowledgements

This work was financially supported by BIOMIN GmbH, Austria.